

to spread in the direction of least resistance from the surrounding harder structures as it becomes larger, making the cyst walls extend like partitions. Therefore, we think that MGEC form from obstructed or occluded follicles in the same way as common epidermal cysts.⁸

Preoperative imaging suggested a cystic lymphangioma or neurilemmoma in patient 1 and a ganglion or a neurofibroma with extensive degeneration in patient 4 (Fig. 1b,h). These lesions may be distinguished by macroscopic findings during surgery. Among the types of multilobulated cysts lined with keratinized stratified squamous epithelium, proliferative epidermoid cysts (PEC) and dermoid cysts (DC) that become large and multilobulated are the lesions that should be distinguished.^{1,8} PEC are locally aggressive tumours, and are potentially malignant. MGEC were distinguished from PEC by the following features. In MGEC, the cyst walls were all composed of nonatypical keratinocytes with normal cellularity and flattening rather than proliferation. Epithelial proliferation from the cyst wall projecting into the lumen or peripherally into the surrounding dermis was not seen in MGEC (Fig. 2). In contrast to MGEC, DC include hair follicles, sebaceous and sweat glands in the epithelium.¹ An epidermal cyst can be diagnosed clinically when there is a characteristic black, central keratin-filled punctum on the epidermis. A connection with the epidermis was observed in all cases, but the extent of tethering varied. However, a central keratin-filled punctum was not observed in any of our patients. This made the diagnosis of epidermal cyst more difficult.

Although our case series comprises only eight patients, half of them had multilocular cysts. Therefore, we now think that giant epidermal cysts are more often multilocular than unilocular. The conventional concept that an epidermal cyst is a unilocular mass may lead to an incorrect preoperative diagnosis of giant epidermal cyst. It must be remembered that a giant epidermal cyst may present as a multilocular lesion on physical examination and imaging.

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Contact urticaria to play dough: a possible sign of dietary allergy

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SIR, Contact urticaria to foods is common in atopic children and is used as a marker of dietary allergy. We report a 3-year-old atopic boy with contact urticaria to red play dough. Open patch testing with coloured play doughs, foods and antibiotic syrups gave contact urticarial reactions to E124 (ponceau 4R, cochineal red A) and E129 (allura red AC). These widely used red colourings have not previously been reported to cause contact urticaria.

A 3-year-old boy developed swollen, erythematous, itchy hands and forearms at nursery while playing with play dough. This rash took 2 h to subside and there was no flaring of his eczema. His mother established that the rash only occurred with the red-coloured play dough. Because of a possible allergy to red dyes in foods and medicines, open patch testing was performed to try to identify the specific allergen. Controls were tested to exclude nonimmunological urticaria.

The manufacturers of coloured play doughs, antibiotic syrups and Smarties® were contacted regarding their product ingredients. The relevant results are shown in Table 1. The

Table 1. Contact urticarial reactions to coloured products tested

Product	Colouring	Contact urticaria 10 min	Contact urticaria 20 min	Contact urticaria 40 min
Red Playdo®	E124, E110	–	+	++
Orange Playdo®	E124, E102	–	–	+
Yellow Playdo®	E104	–	–	–
Red nursery play dough	E124	–	–	+
Red Play Stuff®	E124	–	–	+
Red Smartie®	E124, E122	–	–	+
Orange Smartie®	E110	–	–	–
Pink Smartie®	E120	–	–	–
Green Smartie®	E104, E133	–	–	–
Red cefaclor	E129	–	+	+
Orange penicillin	E124, E110	–	–	–
Yellow amoxicillin	E104	–	–	–

– No reaction; + palpable erythema; ++ palpable erythema, weal and flare.

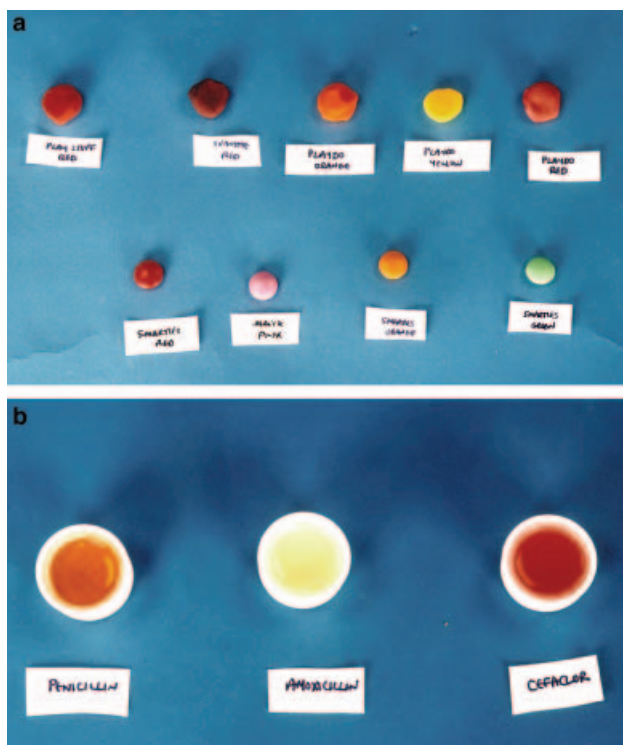


Figure 1. (a,b) Products used for open patch testing.

child was tested to the products shown in Figure 1a,b. Smartie®-sized play dough pieces and Smarties® were applied to the child's forearms under Micropore® adhesive tape. Cotton wool soaked in the antibiotic syrups was applied to the child's back under Micropore® adhesive tape and all sites were reviewed at 10-min intervals. Six control children and six control adults were tested to identical products.

As shown in Table 1, all items containing E124 or E129 produced contact urticaria in the case child, except for orange penicillin in which the red colourant was diluted. The most obvious contact urticarial reaction was seen with red Playdo® (Fig. 2).

The child was given chlorpheniramine syrup and all reactions settled within an hour without flaring of his eczema. Due to the possibility of E124 and E129 causing the oral allergy syndrome, we advised avoidance of these additives both topically and orally.

Contact urticaria [nonimmunological (NICU) or immunological (ICU) in aetiology] is a weal and flare reaction appearing within minutes to an hour after cutaneous exposure to a substance. The prevalence is unknown. Mild reactions show only redness and itching but no weals. NICU causes mild localized reactions in nonsensitized individuals. ICU is an IgE-mediated type I hypersensitivity reaction that occurs after sensitization by cutaneous, oral or inhalation exposure and is more common in atopic individuals. At subsequent exposures the allergen will cross-link mast cell membrane-bound IgE antibodies causing degranulation and



Figure 2. Contact urticarial reaction to red Playdo®.

the release of both preformed and newly synthesized inflammatory mediators.¹ Generalized urticaria, asthma and anaphylaxis, the 'contact urticaria syndrome', can occur after local contact.² The term 'oral allergy syndrome' is now used to describe ICU of the oropharyngeal mucosa.³

NICU can be caused by a variety of products including additives to foods, medicines and cosmetics, such as the preservatives E200 (sorbic acid) and E210 (benzoic acid).⁴ Similarly, several products, particularly foods, produce ICU.^{5,6} Contact urticaria in response to colourings is far less common and has been reported with E102 (tartrazine), E123 (amaranth) and E110 (sunset yellow).⁷ This is the first report of contact urticaria to E124 and E129, which are widely used in foods, medicines and play products. The exact mechanism causing contact urticaria to colourings is not known, but as none of the control children or adults reacted to identical products, it would suggest that our patient had an ICU to E124 and E129. There are few data specifically relating to contact urticaria, allergy or intolerance due to food additives. A multicentre study of 335 atopic children

determined the incidence of intolerance to food additives to be 2%.⁸

The clinical history is important in diagnosing contact urticaria, the oral allergy syndrome and food allergy. Open or closed patch testing, the radioallergosorbent test and the skin prick test are useful for further investigations. The skin application food test (SAFT: gauze impregnated with standardized amounts of food fixed to the skin) was developed for young children to avoid the use of needles. We used a modified SAFT in our case. In 64 children under 4 years of age the SAFT showed excellent correlation with the double-blind placebo-controlled food challenge, which is considered the 'gold standard' investigation for IgE-mediated food allergy.⁹ This suggests that the SAFT is a reliable test for food allergy in young atopic children. Therefore the positive modified SAFT in our patient indicates that a reaction would probably occur on food challenge.

The use of food additives in preparations for infants aged under 36 months is prohibited under European Union regulations, and colourless antibiotic and antihistamine syrups should always be prescribed to young children. A history of urticaria following contact with play dough should be taken seriously because it may forewarn of a dietary allergy.

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Cutaneous metastatic malignant mixed mullerian tumour mimicking cutaneous leiomyosarcoma: a case report

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SIR, Leiomyosarcoma is a malignant neoplasm of smooth muscle cells that may arise from any organ containing smooth muscles, such as the heart, intestine, uterus or bladder. The skin also contains smooth muscle cells in arrector pili muscles and blood vessel walls, and is one of the most frequent sites for its development.¹ Most leiomyosarcomas found in the dermis or subcutaneous tissues are primary tumours developing in the skin. Multiple leiomyosarcomas in the skin, on the other hand, may be considered as metastatic tumours.² However, metastatic leiomyosarcomas in the skin are rare and only a few cases have been reported in the literature.^{3–5}

In June 2003, a 43-year-old woman with a papule on her scalp visited the Department of Dermatology, Yonsei University Medical Centre (Seoul, Korea). Previously, in June 2002, she had been diagnosed as having a malignant uterine tumour with metastasis to the lung. She had been treated by surgical excision of the primary uterine tumour, followed by chemotherapy. At the time of presentation, the chemotherapy had been stopped temporarily as the pulmonary metastatic lesions had reduced in size and her oncologist wanted her general condition to improve. The scalp lesion had developed 2–3 months prior to presentation and was a slow growing, asymptomatic, erythematous hard papule 7 mm in diameter. Lymphadenopathy was not detected, other cutaneous findings were unremarkable, and her general condition appeared to be good.

Skin biopsy showed bundles of spindle-shaped cells mixed with the remaining normal smooth muscle cells, the arrector pili muscles, throughout the entire dermis. The tumour cells had wavy eosinophilic cytoplasm with vacuoles and plump, pleomorphic and hyperchromatic nuclei, indicating cellular atypia. Atypical mitotic cells and giant cells with bizarre nuclei were also observed (Fig. 1). On immunohistochemical staining, the tumour cells were positive for actin, and negative for cytokeratin, S-100 protein, desmin and CD34.

The patient's medical records revealed that her primary uterine tumour was a malignant mixed mullerian tumour (MMMT) composed of 5% endometrial and 95% leiomyosarcomatous components. The histopathological features of the uterine sarcomatous component were identical to those of the scalp lesion (Fig. 2) and included positivity for actin. We thus diagnosed the scalp lesion as metastatic cutaneous MMMT, leiomyosarcomatous component.

The patient was transferred back to the oncology department. In subsequent imaging studies, multiple newly developed metastatic lesions were detected throughout the entire body including the skull, spine and lungs. In spite of intensive chemotherapy, she died 5 months later.